WHAT IS CLAIMED IS:

1. A method of preventing a complication of a primary disorder in patients wherein said complication results from oxidative damage resulting from the generation of reactive oxygen species by AR-NOX, which comprises:

administering to a patient having said primary disorder, in an amount effective to prevent said complication, one or more ubiquinones in a pharmaceutically acceptable carrier.

2. A method for preventing secondary disorders in patients having a primary disorder that causes oxidative damage resulting from the generation of reactive oxygen species by AR-NOX, which comprises:

administering to a patient having a primary disorder, in an amount effective to prevent said secondary disorder, one or more ubiquinones in a pharmaceutically acceptable carrier.

3. The method of claim 1 or 2 wherein the total daily amount of ubiquinone administered is from about 1 to about 500 mg of a composition comprising ubiquinones.

4. The method of claim 3 wherein the total daily amount of ubiquinone administered is from about 1 to about 100 mg of the composition comprising ubiquinones.

5. The method of claim 1 or 2 wherein the ubiquinone is coenzyme Q_{10}.

6. The method of claim 1 or 2 wherein said ubiquinone is not coenzyme Q_{10}.

7. The method of claim 1 or 2 wherein coenzyme Q_{10} is administered with a ubiquinone selected from a group consisting of coenzyme Q_6, coenzyme Q_7, coenzyme Q_8, or coenzyme Q_9.

8. The method of claim 1 or 2 wherein the primary disorder is old age, rheumatoid arthritis, arthritis associated with age, or fatigue associated with age.

9. The method of claim 1 or 2 wherein the primary disorder is a result of aged cells.

10. The method of claim 1 or 2 wherein the primary disorder is cancer.

11. The method of claim 1 or 2 wherein the primary disorder is selected from a group comprising myocardial infarction, alcoholism, favism, malaria, sickle cell anemia, Fanconi's anemia, protoporphyrin photo-oxidation, nutritional deficiencies, Kwashiorkor, thalassemia, dietary iron overload, idiopathic hemochromatosis, metal ion-mediated nephrotoxicity, aminoglycoside nephrotoxicity, autoimmune nephrotic syndromes, oral iron poisoning, endotoxin liver injury, free fatty acid-induced pancreatitis, nonsteroidal antiinflammatory drug induced gastrointestinal tract lesions, glomerulonephritis, autoimmune diseases, vasculitis, hepatitis B virus, Parkinson's disease, neurotoxins, allergic encephalomyelitis, potentiation of traumatic injury, hypertensive cerebrovascular injury, vitamin E deficiency, adriamycin cardiotoxicity, Keshan disease, selenium deficiency, alcohol cardiomyopathy, photic retinopathy, occular hemorrhage, cataractogenesis, degenerative retinal damage, amyotrophic lateral sclerosis, age-related macular degeneration, diabetes, atherogenesis, and atherosclerosis.

12. A method for screening for agents that sequester AR-NOX, comprising:

(a) incubating in a reaction comprising AR-NOX and a test agent for a time sufficient to allow the test agent to bind AR-NOX; and

(b) detecting in the reaction the presence of a complex comprising AR-NOX and the test compound.

13. The method of claim 12 wherein the test agent is detectably labeled by a dye, an enzyme, an isotope, a fluorescent group, or a luminescent group.

14. The method of claim 12 which further comprises incubating AR-NOX in the presence of a positive control that is known to interact with AR-NOX.

15. The method of claim 14 wherein the positive control is ubiquinone.

16. The method of claim 12 wherein the method of screening takes place within a cell.

17. A method of screening for agents that sequester AR-NOX comprising:

(a) incubating in a reaction comprising a mixture of AR-NOX, a test agent, cytochrome c, and a substrate capable of generating reactive oxygen species for a time sufficient for cytochrome c reduction; and

(b) detecting the presence of reduced cytochrome c in the presence or absence of the test compound.

18. The method of claim 17 wherein the compound capable of generating reactive oxygen species is superoxide dismutase.

19. The method of claim 17 wherein the detection of cytochrome c is measured by comparing the measure of spectrophotometric absorbance of the mixture at about 540 nm to 550 nm.

20. A method of screening for agents that sequester AR-NOX comprising

(a). incubating in a reaction comprising a mixture of AR-NOX, a test agent, and a substrate, wherein said substrate is reduced by AR-NOX, for a time sufficient for AR-NOX to reduce said substrate; and

(b). detecting the presence of reduced substrate in the presence or absence of the test compound.

21. The method of claim 20 wherein the substrate is an ascorbate radical.

22. The method of claim 21 wherein the detection of ascorbate radical is measured by comparing the measure of spectrophotometric absorbance of the mixture at about 265 nm.

23. The method of claim 20 wherein the substrate is NAD^+.

24. A method of screening for agents that sequester AR-NOX comprising

(a). incubating in a reaction comprising a mixture of AR-NOX, a test agent, and a substrate, wherein said substrate undergoes disulfide-thiol interchange activity in the presence of AR-NOX, for a time sufficient for AR-NOX to reduce said substrate; and

(b). detecting the presence of disulfide-thiol interchange in the substrate in the presence or absence of the test compound.

25. A method of preventing a complication of a primary disorder in patients wherein said complication results from oxidative damage resulting from the generation of reactive oxygen species by AR-NOX, which comprises:

administering to a patient having said primary disorder, in an amount effective to prevent said complication, an agent that sequesters AR-NOX, identified by the methods of claims 12, 17, 20, or 24, in a pharmaceutically acceptable carrier.

26. A method for preventing secondary disorders in patients having a primary disorder that causes oxidative damage resulting from the generation of reactive oxygen species by AR-NOX, which comprises:

administering to a patient having a primary disorder, in an amount effective to prevent said secondary disorder, an agent that sequesters AR-NOX, identified by the methods of claims 12, 17, 20, or 24, in a pharmaceutically acceptable carrier.

27. The method of claim 25 wherein the primary disorder is old age, rheumatoid arthritis, arthritis associated with age, or fatigue associated with age.

28. The method of claim 26 wherein the primary disorder is old age, rheumatoid arthritis, arthritis associated with age, or fatigue associated with age.

29. The method of claim 25 wherein the primary disorder is selected from a group comprising cancer, myocardial infarction, alcoholism, favism, malaria, sickle cell anemia, Fanconi's anemia, protoporphyrin photo-oxidation, nutritional deficiencies, Kwashiorkor, thalassemia, dietary iron overload, idiopathic hemochromatosis, metal ion-mediated nephrotoxicity, aminoglycoside nephrotoxicity, autoimmune nephrotic syndromes, oral iron poisoning, endotoxin liver injury, free fatty acid-induced pancreatitis, nonsteroidal antiinflammatory drug induced gastrointestinal tract lesions, glomerulonephritis, autoimmune diseases, vasculitis, hepatitis B virus, Parkinson's disease, neurotoxins, allergic encephalomyelitis, potentiation of traumatic injury, hypertensive cerebrovascular injury, vitamin E deficiency, adriamycin cardiotoxicity, Keshan disease, selenium deficiency, alcohol cardiomyopathy, photic retinopathy, occular hemorrhage, cataractogenesis, degenerative retinal damage, amyotrophic lateral sclerosis, age-related macular degeneration, diabetes, atherogenesis, and atherosclerosis.

30. The method of claim 26 wherein the primary disorder is selected from a group comprising cancer, myocardial infarction, alcoholism, favism, malaria, sickle cell anemia, Fanconi's anemia, protoporphyrin photo-oxidation, nutritional deficiencies, Kwashiorkor, thalassemia, dietary iron overload, idiopathic hemochromatosis, metal ion-mediated nephrotoxicity, aminoglycoside nephrotoxicity, autoimmune nephrotic syndromes, oral iron poisoning, endotoxin liver injury, free fatty acid-induced pancreatitis, nonsteroidal antiinflammatory drug induced gastrointestinal tract lesions, glomerulonephritis, autoimmune diseases, vasculitis, hepatitis B virus, Parkinson's disease, neurotoxins, allergic encephalomyelitis, potentiation of traumatic injury, hypertensive cerebrovascular injury, vitamin E deficiency, adriamycin cardiotoxicity, Keshan disease, selenium deficiency, alcohol cardiomyopathy, photic retinopathy, occular hemorrhage, cataractogenesis, degenerative retinal damage, amyotrophic lateral sclerosis, age-related macular degeneration, diabetes, atherogenesis, and atherosclerosis.

31. The method of claim 25 wherein a response to the agent that sequesters AR-NOX is monitored in sera.

32. The method of claim 26 wherein a response to the agent that sequesters AR-NOX is monitored in sera.

33. The method of claim 31 wherein AR-NOX is detected with an antibody specific to AR-NOX.

34. The method of claim 32 wherein AR-NOX is detected with an antibody specific to AR-NOX.

35. The method of claim 33 wherein the antibody is conjugated to a label wherein the label provides a detectable signal.

36. The method of claim 34 wherein the antibody is conjugated to a label wherein the label provides a detectable signal.

37. The method of claim 31 wherein AR-NOX is detected by an enzymatic assay.

38. The method of claim 32 wherein AR-NOX is detected by an enzymatic assay.

39. The method of claim 37 wherein the assay is reduction of cytochrome c.

40. The method of claim 38 wherein the assay is reduction of cytochrome c.

41. The method of claim 37 wherein the assay is reduction of an ascorbate radical.

42. The method of claim 38 wherein the assay is reduction of an ascorbate radical.

43. The method of claim 37 wherein the assay is oxidation of NADH.

44. The method of claim 38 wherein the assay is oxidation of NADH.

45. The method of claim 37 wherein the assay is measuring disulfide-thiol interchange activity.

46. The method of claim 38 wherein the assay is measuring disulfide-thiol interchange activity.

47. The method of claim 31 wherein AR-NOX is detected by measuring an oscillation rate of AR-NOX.

48. The method of claim 32 wherein AR-NOX is detected by measuring an oscillation rate of AR-NOX.

49. The method of claim 31 wherein AR-NOX is detected by measuring its resistance to retinoic acid.

50. The method of claim 32 wherein AR-NOX is detected by measuring its resistance to retinoic acid.

51. The method of claim 31 wherein AR-NOX is detected by identifying cells with depressed mitochondrial functions.

52. The method of claim 32 wherein AR-NOX is detected by identifying cells with depressed mitochondrial functions.

53. A pharmaceutical composition comprising a compound that inhibits the generation of reactive oxygen species by AR-NOX in a cell that is identified by the methods of claims 12, 17, 20, or 24, and a pharmaceutical acceptable carrier.

54. The composition of claim 53 wherein the composition is formulated as a capsule, tablet, soft gel, solution, suppository, injection, aerosol, or a kit.